Gases(M.Mortada)

<table>
<tr><th></th><th>Carbon Monoxide(CO)(The silent Killer; The invisible Killer)</th><th>Cyanide poisoning</th></tr>
<tr><td>Definition</td><td>CO is a gas produced as a product of incomplete combustion of carbonaceous materials.</td><td>- one of the most rapidly acting lethal poisons. It can be a gas, liquid or solid and may exhibit a faint bitter almond like odor.</td></tr>
<tr><td>Physical</td><td>carbon monoxide is referred as silent killer because it is odorless, colorless, tasteless and non-irritating gas</td><td></td></tr>
<tr><td>Sources</td><td>1- incomplete combustion (burning) of any carbonaceous fuel.
2- incomplete combustion of natural gas (methane) (natural gas itself does not contain CO).
3- Motor vehicle exhaust.
4-charcoal grills when used in closed poorly ventilated areas.
5- Fires are important source of CO exposure.
6- Methylene chloride, a paint removers is another source of co. It's readily absorbed by ingestion, inhalation and through Skin. It is metabolized by liver into CO.</td><td>1- Ingestion of cyanide salts (sodium or potassium cyanide) or Inhalation of hydrogen cyanide gas.
2-Naturally occurring cyanogenic glycoside amygdalin in certain nuts, plants& fruit pit (apricot, peach, bitter almond &cherry)
→when ingested :amygdalin is bio transformed by intestinal glycosidase to cyanide.
3-Ingestion of agents that are metabolized to cyanide such as (acetonitrile and acrylonitrile)which are themselves non toxic but biotransformation via CYP450 liberates cyanide.</td></tr>
<tr><td>Mode of toxicity</td><td>1- Accidental exposures from home heating, smoke inhalation and automobile exhaust.
2-homicide
3- Suicide.</td><td>1- Non intentional (accidental) exposures:
• In chemists or technicians working in laboratories where cyanide salts are common reagents.
• Following smoke inhalation.
• Iatrogenic cyanide poison during use of nitroprusside as a VD to↓bl. pr(each nitroprusside molecule contain 5 molecules cyanide).
2- Intentional for suicide by ingestion of potassium cyanide(KCN).
Lethal dose:60- 90 mg Hydrogen Cyanide (HCN)&200 mg Potassium Cyanide (KCN)
Fatal Period : (10 Min)</td></tr>
<tr><td>Mech of toxicity</td><td>CO binds to Hb to form carboxyhemoglobin (CO Hb); which render Hb incapable of delivering O_2 to cells .
It has affinity for Hb 200-250 times greater than that of oxygen.
The toxic effects of CO result from tissue hypoxia which is due to:
a) ↓ amount of O_2bound to erythrocytes. Thus causing a reduction in tissue oxygenation.
b) Shifting of Oxy Hb dissociation curve to left, lowering the partial pr at which O_2 is available to the tissues.
®The organs most sensitive to hypoxia are: brain &heart; but with ↑ Severe exposure, other organs are Susceptible to CO effects.</td><td>- Cyanide has a high affinity for metals, forming complexes with metallic cations.
- It binds to the ferric (Fe^{+3}) of mitochondrial cytochrome oxidase→inhibiting its function.
- It disrupts the ability of cells to use O_2, resulting in tissue anoxia,↑anaerobic metabolism and the rapid development of acidosis.</td></tr>
<tr><td>P/K</td><td>Abs:CO is readily inhaled and absorbed at a rate proportional to resp exchange rate.
Ms: half- life of CO is 4-5 hr.It ↓ to 80 min e' 100% O_2 and to 23 min e' hyperbaric oxygen.
Ex:CO is eliminated from the lungs unchanged.</td><td>Abs: rapidly absorbed by Resp Tract and MM and more slowly by GIT and the skin.
Ds: rapidly into RBCs &tissues.
MS: Detoxification through combination of cyanide e' sulfur to form thiocyanate→which is eliminated by the kidney.</td></tr>
<tr><td>C/P</td><td>CO exposure tends to be Seasonal, with most exposures in the winter.
VIQ Symptoms&signs at various CO Hb Concentarations:
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<tr><th>COHb%</th><th>Signs and symptoms</th></tr>
<tr><td>0 – 10%</td><td>No symptoms (asymptomatic)</td></tr>
<tr><td>10-20%</td><td>Tightness across forehead, slight headache, excertional dyspnea & dilatation of cutaneous bl.vs</td></tr>
<tr><td>20 - 30%</td><td>Headache, throbbing in temples , easy fatigue & dizziness</td></tr>
<tr><td>30-40%</td><td>Severe headache, weakness, dizziness, confusion, diminution of vision, nausea, vomiting & collapse</td></tr>
<tr><td>40-50%</td><td>Same as above+ collapse, syncope and ↑↑pulse and respiratory rate.</td></tr>
<tr><td>50-60%</td><td>Syncope,↑↑pulse and respiratory rate ,coma, intermittent convulsions and Cheyne- Stokes respiration.</td></tr>
<tr><td>60 – 70%</td><td>Coma, intermittent convulsions, ↓↓ pulse & respiratory rate and possibly death.</td></tr>
<tr><td>70-80%</td><td>Weak pulse, respiratory failure and death within few hours.</td></tr>
<tr><td>80- 90%</td><td>Death in less than an hour.</td></tr>
<tr><td>> 90 %</td><td>Death within few minutes.</td></tr>
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Cherry-red coloration of the skin has been attributed to CO, seen in the end stage.

Delayed effects:
→ involve lesions of the cerebral white matter & basal ganglia.
C/P→dementia, amnestic syndromes, psychosis, parkinsonism ,paralysis, chorea, cortical blindness, peripheral neuropathy &urinary incontinence)</td><td>*The severity of symptoms depends on the actual amount and rate of cyanide exposure.
* The onset of symptoms is immediate after inhalation.
* Symptoms begin within minutes after the ingestion of cyanide salts.
* Symptoms are delayed after the ingestion of compounds that require metabolic activation.
Symptoms and signs:
1- CNS: Headache, drowsiness, dizziness, seizures and coma.
2- CVS: HTN, tachycardia, hypotension, bradycardia and asystole.
3- Resp: Dyspnea, tachypnea & apnea
4- After oral ingestion of cyanide salts:
• Smell of bitter almonds from mouth.
• Burning of the tongue and mucous membranes.
• GIT irritation.</td></tr>
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<td>Diagnosis</td>
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- History: of exposure to any source of CO.
- C/P : as before.
- Lap investigations:
1) Carboxyhemoglobin (CO Hb) level:
- most important lab to confirm CO poisoning.
- Normal levels are less than 5% in non-smokers &c high as 12% in two pack-per-day smokers.
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<tr><th>A-Qualitative assessment</th><th>B-Quantitative assessment</th></tr>
<tr><td>a) Dilution test
b) Kunkel's test: Rapid field test & only qualitative.
A few drops (5 to 6 drops) of 3% tannic acid add to patient's diluted blood 1 :10 with distilled water gives a persistent crimson red coagulum, indicates the presence of CO Hb.
c) Spectroscopic examination.</td><td>a) Spectrophotometer.
b) Gas chromatography</td></tr>
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2) An immediate bed side determination of blood glucose.
3) Cardiac monitoring should be initiated and an ECG obtained.
4) ABG study is essential.
5) Electrolytes will confirm metabolic acidosis
6) CBC &renal function tests
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<td>
-History: of ingestion or exposure to cyanide.
- C/P: as before.
- Lap investigations:
1- ABG → confirm gap metabolic acidosis.
2- Serum electrolytes
3- Serum cyanide level: →
*Toxic > 0.5 mg /L
*Fatal > 3.0 mg /L
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<td>DD</td>
<td>CO poisoning is most commonly misdiagnosed as influenza, food poisoning, gastroenteritis and even colic in infants.</td>
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<td>TTT</td>
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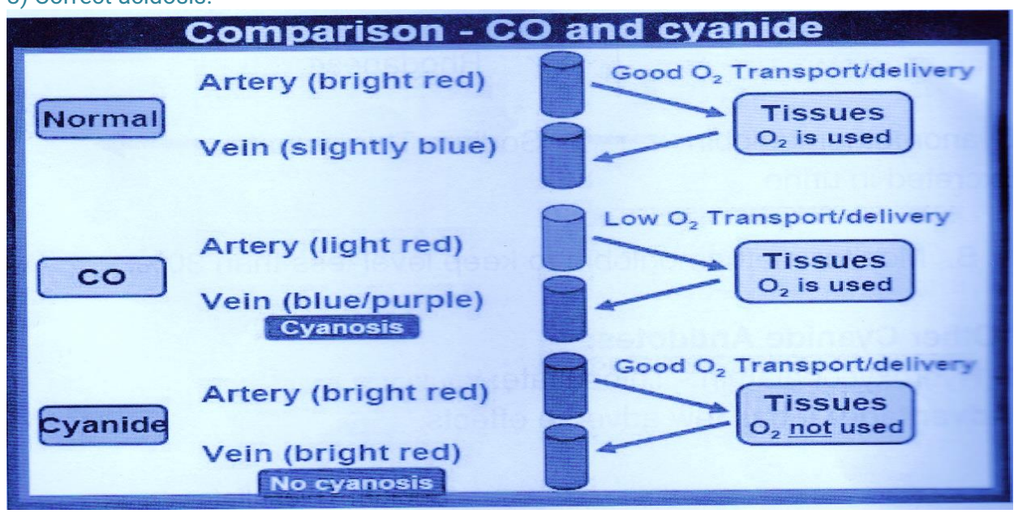
1) Remove patient from source of exposure.
2) initial ttt is directed at providing immediate oxygen(100 % O_2) through face mask should be used to →
- Promote cellular respiration.
- Reduce the elimination half-life of CO Hb from 4 - 5 hrs to 1- 2hrs.
3) Hyperbaric Oxygen (HBO):
oxygen at a pr about 2-3 times that of normal air pressure →
*Improved tissue oxygenation.
*Enhanced elimination of CO Hb.
*Improve cerebral edema.
ttt with oxygen should continue until the patient is asymptomatic(usually when CO Hb level is less than 10 %).
4) For cerebral edema, give cerebral dehydrating agents as corticosteroids or mannilol 20% .
5) cardiac dysrhythmias should be ttt e' appropriate antiarrhythmics.
6) Care of coma and ttt of pulmonary edema.
7) Warm the patient.
8) Correct acidosis.


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I- General measures:ABCD
A Evaluate adequacy of air way.
B Assess adequacy of oxygenation (administer 100 % oxygen)& ventilation.
C Establish IV access.
Maintain blood pressure via fluids and vasopressor agentsis necessary.
Cardiac dysrhythmias should be treated with appropriate antiarrhythmics.
D Seizures treated with diazepam and phenytoin.
II- Decontamination:
a) Respiratory: Remove the patient from site of exposure.
b) Oral: GL e' cuffed end tracheal intubation is indicated in patients e' recent cyanide ingestion.
Activated charcoal binds small amounts of cyanide and may↓significance of ingestion especially recent ingestion.
II- Antidotes (Toxin specific measures) →1) Cyanide Antidote Kit (Nitrite -thiosulfate):
-contains amyl nitrite, sodium nitrite & sodium thiosulfate.
-Nitrites induce methemoglobin.so It causes removal of cyanide from tissues by the high affinity of cyanide to bind metHb
• Step 1 : Amyl nitrite (inhalation until IV line established)
• Step 2 : sodium nitrite (10 ml{300mg} of 3 % IV over 5-10 mins).
Nitrate Cyanide-cytochrome enzyme
Hb(Fe^{+2}) MetHb(Fe^{+3}) CyanometHb
• Step 3: Sodium thiosulfate(50ml{12.5g} of 25% IV over 20 mins)
Na^{+}thiosulphate
CyanometHb sodiumThiocyanate Excrete in urine
Rhodnase
N. B: Monitor methemoglobin to keep level less than 30%.
2)Other Cyanide Antidotes:
1 - Hydroxycobalamin - Thiosulfate:
cobalmin(recycle again)
Na^{+}thiosulphate
Hydroxycobalamin+ cyanide Cyanocobalmin(Vit B12) sodiumThiocyanate Excrete in urine
Advantage: It has few adverse effects. Rhodnase
Concurrent add thiosulfate to recycle the hydroxycobalamin binding and may ↓amount of hydroxycobalamin require to detoxify cyanide.
2- Dicobalt EDTA (Kelocyanor):
Advantage: It chelates cyanide by forming stable complex with cyanide; as cobalt compounds have a high affinity for cyanide.
Disadvantage: It has significant toxicity in the absence of cyanide(misdiagnosis of cyanide).
3-Di methylami nophenol (DMAP) :
Advantage: A rapid methemoglobinemia forming agent.
Disadvantage:*Severe hypotension →due to sodium nitrite.
* Associated with renal failure in experimental animals.
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